January 3, 2008
2858 Pheasant Lane
Stevensville, MT 59870

Dear County Commissioners,

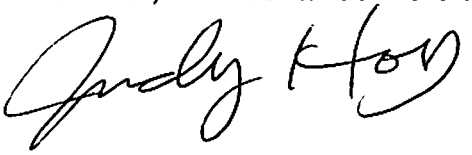
I sincerely hope that all of you will read this review of causes of Congenital Fetal Hypothyroidism and the connection to the symptoms in young vertebrates born in Ravalli County, Montana. The symptoms of Congenital Fetal Hypothyroidism (CFH) completely match the symptoms in wild and domestic animals that we have been documenting for 12 years. This review represents 12 years of hard work as a public service (for no pay) by many Ravalli County citizens. Our pay will be to have viable, healthy young born and hatched.

Some who have read the review question whether Chlorothalonil or its nitrile metabolites are involved in causing the epidemic levels of CFH here in Western Montana, since not much Chlorothalonil is used here. I concede that point and Chlorothalonil's involvement needs to be proven with more tests. If Chlorothalonil and other chemicals not used here are not involved in the epidemic of CFH symptoms, that means that we are totally to blame for the hundreds of thousands of young vertebrates that have been born with those symptoms in Ravalli County during the last 12 years, including human newborns. That also means that we are going to have to mitigate the known causes listed in the review or the air, water and plants in the Bitterroot Valley will become too toxic to support all life. Those necessities appear to now be nearing maximum toxicity for wild ungulate fetuses and newborns, for amphibian and reptile young and likely the young of some species of birds (those in decline for no observable reason, because the young do not survive to be observed).

The Commissioners are empowered to protect the health, welfare, safety and property of the citizens of the county. It appears that the first three of those have fallen by the wayside. Actually, all the malformed foals, calves, goats, sheep, llamas, domestic fowl and pets that have symptoms of CFH are also citizens' property. Obviously that property is not being protected.

Thank you for your very good recent efforts to make Ravalli County a safer and healthier place to live. However, we must all do more and soon.

Sincerely,
Judy Hoy



P.S.
This is for the Commissioners who have repeatedly stated that what we have documented are normal variations or are not really congenital developmental malformations. The rest of you can skip this part. The scientific journal and the scientists who reviewed our 2002 study of underdeveloped and misaligned genitalia verified those problems as congenital developmental malformations. Top veterinarians from states other than Montana have confirmed that underdevelopment of the skull, upper face, palate, with resultant underbite is not considered a normal variation by any "respectable veterinarian" unless the animal is deliberately bred to be that way. Do you know that the ranchers and the Montana Department of Fish, Wildlife and Parks are deliberately breeding the wild and domestic ungulates to have those characteristics? We would assume that no one would want a grazing animal to be born with underbite, so why the cover-up of the problem?

A Review of Studies That Indicate The Causes of Congenital Fetal Hypothyroidism and Associated Adverse Health Effects, Including the Definitive Congenital Developmental Malformations That Appear To Be Epidemic in Perinatal Vertebrates in Western Montana

Judy Hoy, 2858 Pheasant Lane, Stevensville, MT 59870 Phone - 406-777-2487 December, 2007

For 11 years, we have been documenting birth defects on vertebrate species in Western Montana. Underdevelopment of skull and facial bones, and underdevelopment or maldevelopment of the external genitalia (on the outer skin) are the two congenital malformations most observed on our primary study animal, white-tailed deer. We now know that thyroid gland function is being seriously compromised, called Congenital Fetal Hypothyroidism (CFH), on vertebrate embryos and fetuses, resulting in the above and other birth defects documented on young of many species of mammals and birds;.

Precisely the same symptoms of cranial-maxillary underdevelopment and maxillary brachynathia (underbite because of short upper face - commonly referred to as prognathism), as well as most of the other birth defects we have observed on ungulates here in Montana was reported in a study by Andrew Lyndon Allen (A.L. Allen, Investigations into Congenital Hypothyroidism of Foals, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, October, 1996). The same fetal malformations, including prognathism, were reported in a study of goats that lived in an area of very low iodine (J.L. Singh, M.C. Sharmar, Mahesh Kumar, V.P. Varshney, A.H. Ahmad and Shiv Prasad, Clinico-biochemical profile and therapeutic management of congenital goitre in kids, Indian J. Vet. Med. Vol. 23, No.2, 2003 pp. 83-87) and most importantly, in another study of goats, deliberately dosed with cyanide (Soto-Blanco and Gorniak, Prenatal toxicity of cyanide in goats -- a model for teratological studies in ruminants, *Theriogenology*, 62 1012-1026, 2004.) Goats are the domestic ungulate species here in Ravalli County with the highest rates of symptoms of CFH. In each year immediately prior to having high rates of maxillary brachynathia, Western Montana animals were exposed to significant amounts of smoke from wildfires that included house fires, releasing large amounts of cyanide into the smoke. Additionally, millions of pounds of Chlorothalonil applied in states to the west of Ravalli County metabolizes into nitrile (cyanide) metabolites that come here to Western Montana on weather fronts. Rain and snow that contain the Chlorothalonil, metabolites (nitriles) and a contaminant, Hexachlorobenzine (banned by the EPA), as well as many other toxins applied or released into the air here and in states to the west, fall on the foliage and into the surface water that are then ingested by ungulates.

The key is that Chlorothalonil is more soluble than most chlorinated pesticides. Being more soluble in water, Chlorothalonil is more soluble in the blood and thus more available to interfere in cellular functions. This property makes it just right to replace similar iodine molecules in the cells of the thyroid glands. The thyroid glands need iodine to function properly. Functional hypothyroidism results when the thyroid is not able to produce the proper amount of thyroid hormones, T4 and T3, because of the replacement of iodine by similar molecules. Proper levels of

thyroid hormones are needed to direct normal growth of brain cells, nerves, bones, muscles, reproductive organs, eyes, ears, the immune system, including the thymus, and other organs on a developing fetus.

According to Dr.Theo Colborn, Dr; Douglas Seba and Dr. Diane Henshel (personal communications), Chlorothalonil is similar to other polyhalogenated aromatic hydrocarbons (PHAHs) that cause multiple deformities and effects on multiple organs, but is even more potent. Chlorothalonil is a benzene ring with a chlorine at four carbons and two cyanides at the other two carbons, a small compound that penetrates the cells of the thyroid gland very efficiently. The cyanide helps the Chlorothalonil be more water soluble without significantly reducing the lipid solubility. Most significantly the cyanides increase reactivity, thus the ability of the fetal thyroid to promote the normal growth of organs is seriously compromised, especially with simultaneous exposure to a variety of thyroid hormone disrupting agents. The fetal organs affected and the severity of the damage depend upon two main factors. One is how many other thyroid hormone disrupting agents or conditions the fetus is being exposed to at the time of a serious Chlorothalonil exposure (mainly via atmospheric transport in our area of Montana). A great many manufactured compounds and some heavy metals produce serious thyroid dysfunction at very low levels of exposure. Second, the timing of a significant thyroid disfunction episode likely dictates the severity of the malformations and/or which organs are affected. The exposure to the cyanide increases the reactivity of all the other agents to which the developing fetus is exposed. This property of cyanide appears to be causing the effects of thyroid dysfunction in vertebrate fetuses to be far more severe since heavy planet wide spraying of Chlorothalonil began, than prior to 1994, when only a small amount was used.

A study of newborn pigs exposed to cyanide by the mothers eating black cherries had similar symptoms to other studies of thyroid hormone disruption and the male piglets had underdeveloped external genitalia (Selby, et al., Case AA. Outbreak of swine malformations associated with the wild black cherry Prunus serotina. Arch Environ Health 1971: 22:496-501.); very important because in 1995, the same spring as the other documented malformations, animals began began being born with extremely high rates of genital malformations. This is a developmental malformation we reported in our study published in 2002 (Hoy, et al., (2002) Genital abnormalities in white-tailed deer (Odocoileus virginianus) in west-central Montana: Pesticide exposure as a possible cause., *J. Environmental Biology*, 23(2), 189-197).

Additionally, Theo Colburn, in her review article published the same year, (T. Colburn, "Clues from Wildlife to Create an Assay for Thyroid System Disruption", published in *Environmental Health Perspectives*, Vol. 110, Supplement 3. (June, 2002), pp. 363-367), discusses many studies of fetal exposure to PCBs, dioxin, pesticides (especially organochlorine compounds), and heavy metals that have produced the same CFH symptoms in prenatal and post natal wildlife, indicating that exposure to those toxins also results in fetal thyroid hormone disruption. Many of the studies that she discusses concern skull and maxillary bone, as well as limb bone malformations in wild and domestic bird species. Hatchling birds of many species with underdevelopment of maxillary bones, limb bones, feathers and/or rhamphotheca have been observed by wildlife rehabbers and bird watchers in Western Montana, beginning spring of 1995.

A list of the characteristics of dysmature foals with CFH are from mild to severe underbite, contracted tendons and the inability to stand, soft silky haircoats (weird fluffy curly

hair on deer and failure of feathers to grow on bird species), soft pliable ears (curled ear tips that don't straighten on deer and antelope - rolled ears on calves), weak tendons, laxity of ligaments around the leg joints, umbilical hernias, poor muscling, and underdeveloped bones (Allen, 1996). Many other studies described similar symptoms of CFH; with all of them stating prognathism as one of the symptoms.

The symptoms listed for foals and other study animals with thyroid hormone disruption are nearly identical to what we have documented in deer, elk, pronghorn antelope, domestic goats, sheep, cattle, horses, llamas, other ungulate species, a few individuals of carnivore species and individuals of many bird species. All of the symptoms of CFH, with the exception of the jaw malocclusions and the reproductive malformations, would cause high mortality in wild animals and thus, we have documented far more wildlife with jaw malocclusion and maldeveloped and/or underdeveloped external genitalia than with any of the other symptoms. All symptoms are reportedly quite common and easily observed on newborn domestic ungulates.

Underbite or maxillary brachynathia, as a result of the underdevelopment of the upper face and skull bones is quite diagnostic; being a lesion that has not been reported as a symptom of other common disease. I have many cleaned skulls of a variety of ungulate species that clearly show that the skull and maxilla are not fully developed and photos of the face and mouth of many of the above listed animals taken in years between 1996 and 2007. Additionally, I have photos of a herniated umbilicus, contracted tendons, hyperextended legs, crooked legs because one side of the bone grew faster than the other, micrognathia (underdeveloped lower jaw), no hair, abnormal hair, curled ears, as well as abnormal feathers and underdeveloped skull and upper bill on bird species. I saw none of these congenital lesions on the hundreds of ungulates I observed in years prior to spring of 1995.

In 2001, the rate of cranial-maxillary underdevelopment with resultant underbite went up significantly in white-tailed deer to slightly over 50% and averages 35% for all years documented. This is far higher than the 5% that is supposed to raise a red flag according to biology textbooks. The hunter killed male elk, mule deer and pronghorn antelope that we have documented had a rate of from 50 to 60 percent in 2005 and 2006 animals examined. Cranial-maxillary underdevelopment and resultant underbite has been verified as a serious congenital developmental malformation by many "knowledgeable scientists," one of whom, Samuel Holland, DVM, said the following concerning a newborn goat with a severely underdeveloped palate and underbite, "-- it seems you're identifying a syndrome in goats that is not readily recognized by several very knowledgeable scientists." He also stated in reply to my inquiry as to whether the malformation could be in any way considered a "normal variation", " The important thing is probably the fact that what you are seeing should not be "normal variation" unless it is an inherited birth defect that some folks have bred into the population and wish to continue this approach!!!" No hunter or livestock owner to whom I have spoken, wants grazing animals to have underbite or an underdeveloped skull. Additionally, Mark Adkins, Breed Standard Chairperson for the National Pygmy Goat Association stated: "The Board kicked around the idea of stopping judges from checking bites at NPGA shows, but the consensus was that the seriousness of malocclusions outweighed the risk of spreading disease."

High rates of underbite in ungulate species indicate a widespread and possibly escalating problem with disruption of the thyroid hormones of developing fetuses. The most common

contaminants that cause CFH are nitrates, cyanide and nitriles, as well as, dioxin, organochlorine compounds and other PHAHs, Mercury, PCBs, and others, or a combination of those toxins. Soil mineral deficiencies also contribute to CFH, as shown by the study of goats in an area of deficient Iodine (Singh, et al., 2003). Selenium deficiency in the soil, such as we have in Ravalli County, interferes with the conversion of T4 to T3, thus would significantly contribute to hypothyroidism in animals exposed to the above toxins.

Higher than normal nitrates in foliage have commonly been shown to result in disruption of fetal thyroid hormones (Allen, 1996). Regarding nitrates, Allen states, "An association between nitrate exposure and alterations in iodine metabolism, thyroid activity or thyroid gland morphology has been reported in a variety of animals including fish, rats, growing pigs, goats, lambs and sheep, and cattle" and "It has also been shown that nitrate is able to cross the placenta of rats, guinea pigs, pigs, and cattle" (Allen, 1996).

The many studies in which nitriles were given to hamsters by Calvin Willhite and colleagues in the 1980's caused fetal and maternal effects. Symptoms noted on hamster fetuses for most nitriles included significant incidence of malformations, including exencephaly, encephalocoele and runting, skeletal malformations and heart damage. The heart damage documented by Willhite is especially pertinent because beginning in fall of 2006, the hearts I examined from all ages of ungulates, both wild and domestic, have had very prominent dilated lymphatic vessels on the surface of the hearts. Many hearts, including those of newborns have an enlarged right ventricle with thin heart wall on that chamber since 1997.

Saillenfait and Sabate found that all the nitriles that they investigated produced the characteristic defects developed by embryos exposed to sodium cyanide *in utero* or in culture. They stated, "Our results provide further evidence that maternal production of cyanide may contribute to the developmental toxicity of saturated and unsaturated nitriles and suggest that distinct metabolites derived from microsomal metabolism of unsaturated nitriles may also play a role." (A. M. Saillenfait1 and J. P. Sabaté, Comparative Developmental Toxicities of Aliphatic Nitriles: *In Vivo* and *in Vitro* Observations, Toxicology and Applied Pharmacology, Volume 163, Issue 2, 1 March 2000). This may be very significant, especially if microsomal metabolism in the digestive system of ungulates is increasing or changing Chlorothalonil or its metabolites to make them more toxic to the thyroid hormones and more disruptive of thyroid hormone function.

Additionally, maternal malnutrition has been been found to disrupt thyroid hormone function and result in less alkaline body fluids. In studies of sheep, the development of male reproductive organs was altered by undernutrition because disruption of thyroid hormones in turn disrupted the pituitary response to gonadotropin-releasing hormone (GnRH) in the male fetuses. (Rae, M.T., S.M. Rhind, C.E. Kyle, D.W. Miller and A.N. Brooks, Maternal undernutrition alters triiodothryonine concentrations and pituitary response to GnRH in fetal sheep, *Journal of Endocrinology* (2002) 173, 449-455.a). This is a very significant finding, in view of the increasing rates of scrotal and penis sheath underdevelopment we are observing on our study animal, white-tailed deer. It is likely that damage to the thyroid by Chlorothalonil in combination with its nitrile (cyanide) metabolites, in addition to all the other thyroid hormone disrupting toxins to which a fetus is now exposed, would also disrupt pituitary response to GnRH. This is even more likely in view of the fact that the underdevelopment of the genitalia began the spring immediately after the huge increase in Chlorothalonil use on potatoes for potato

blight in summer of 1994.

Thyroid dysfunction can be produced if iodine, selenium, and to a lesser extent other minerals are low or absent in the diet or in the soil in which the food plants grow. Inadequate iodine in the soil produced symptoms in the newborn goats, "absence or loss of hair, waddling gait, weakness, letharginess, flexion and enlargement of joints, prognathic face and dome shape appearance of head" (Singh, et al., 2003), which are very similar to those found in the studies of cyanide or nitrate exposure. Inadequate nutrition and/or inadequate minerals are usually factors more influential in wild ungulate fetal growth than with fetuses of well-fed domestic ungulates, especially in areas with cold winters and deep snow or areas with inadequate grazing due to drought.

Exposure to thyroid hormone disrupting toxins, in combination with malnutrition and inadequate selenium and/or iodine deficiency, experienced by many females of RC wild ungulate species in some years, may be a contributing factor to the high rate of external reproductive organ underdevelopment in newborn males of those species in addition to similarly high rates of underbite, skewed sex ratios and other symptoms consistent with CFH in both sexes. Well fed female cattle and sheep raised in RC, given adequate amounts of minerals, reportedly have much lower rates of CFH symptoms than wild ungulates, particularly reproductive underdevelopment and skewed sex ratios according to RC cattle ranchers with whom I have had discussions on this subject.

The fetus is quite resilient, often making up growth lost during a period of maternal malnutrition, when and if the mother acquires adequate food, and thus adequate vitamins and minerals. However, there are likely continuous combined environmental fetal exposures to low levels of thyroid hormone disrupting compounds such as PCBs, PHAHs and others, particularly Chlorothalonil, its nitrile metabolites and cyanide, in addition to heavy metals, radiation, nitrates and other toxins. A great many studies indicate that continuous functional hypothyroidism, especially in combination with several episodes of sudden increased thyroid disfunction, immediately prior to conception and during fetal development appears to result in serious permanent health problems and/or malformations that often result in mortality. This would cause the much lower calf per cow or fawn per doe ratio for which predators are blamed.

Machado, et al.(2001) found a significant correlation between the level of maternal hyperglycemia and the malformation rate in the young. Also, very significantly, the sex ratio for live fetuses in the litters was significantly skewed toward male fetuses (Antonio F. Machado, Ernest F. Zimmerman, David N. Hovland Jr., Robert Weiss, and Michael D. Collins, Diabetic Embryopathy in C57BL/6J Mice : Altered Fetal Sex Ratio and Impact of the Splotch Allele, *Diabetes,* 50:1193-1199, 2001). Our study of RC White-tailed deer found a significantly skewed sex ratio in favor of males with the highest being in 1997 at 66M to 34F per hundred fawns. A large number of studies show that hyperglycemia causes the body fluids to be slightly less alkaline than normal. Also studies have shown that less alkaline maternal body fluids during conception result in the destruction of the XX carrying sperm, leaving only the XY carrying sperm to fertilize the egg or eggs. Thus a highly skewed sex ratio in favor of males would result in a population of maternal females with hyperthyroidism and resultant hyperglycemia. Interestingly, goat owners throughout the U.S. reported an extremely high rate of male birth s in 2003, up to 80%, the same year that Canadian horse owners had up to 80% foal loss due from

CFH.

At present, environmental chemicals that disrupt thyroid hormone function are tested by measuring their ability to affect circulating levels of thyroid hormones (Zoeller, R.T., A.L.S. Dowling, C.T.A. Hertzig, E.A. Iannacone, K.J. Gauger, and R. Bansal, Thyroid hormone, brain development, and the environment, *Environmental Health Perspectives*, Vol. 110, Supplement 3, June 2002.) It is likely that essential thyroid hormone actions, especially in developing young of vertebrates, can be disrupted without detectable changes to thyroid hormone levels as they are currently measured. While sex hormones have been most studied, hormone disrupters that react with thyroid hormone and vitamin A receptors may be even more damaging to fetal growth and health (R.R. Rolland : A review of chemically-induced alterations in thyroid and vitamin A status from field studies of wildlife and fish, J. Wildl. Diseases, 36(4), 15-35, 2000).

A developing fetus is very susceptible to extremely small amounts of molecules of toxins like PHAHs that can cross the placenta, being affected by parts per trillion or even parts per quadrillion, according to studies of effects of hormone disrupting chemicals (Theo Colborn, *Our Stolen Future*, Dutton, 1992). Studies state that the thyroid glands of all vertebrate young, fish, amphibian, reptile, bird and mammal, appear to be similarly affected by such toxins. This would result in the observably similar health problems and malformations in all five families of vertebrates here in Montana. Very high rates of CFH symptoms began being observed in vertebrates throughout the U.S. and in Canada in summer and fall of 1994 and spring of 1995, corresponding exactly in time to the beginning of the abrupt huge increase in use of Chlorothalonil, with applications of hundreds of millions of pounds throughout the Northern Hemisphere.

On examined animals born in spring of 2007 in Ravalli County, including 14 white-tailed deer, 2 mule deer, one elk calf and 9 domestic goat kids, 7 of the white-tailed deer fawns did not have underbite. The rest (19) of the 26 young animals examined had a palate that was short and much narrower than the incisors. The other most common malformation found on those animals was malformed genitalia on 8 of the 8 male white-tailed deer fawns. The rate of males with normal genitalia at the time of our white-tailed deer study was approximately 33% of over 250 animals, with two thirds having a developmental malformation of the genitalia. That rate included a very distinctive malformation that I have not found described in any other study. On many individuals of ungulate species and some individuals of other mammal species (rats, hamsters and dogs have been observed), males are being born with the left hemiscrota formed on the external skin directly anterior to the right hemiscrota. On the developing fetus the left testis descends to a position directly anterior to the right testis, forming a misaligned scrotal sac on the external fetal skin. Such scrotal sacs can not be turned to a normal bilateral position and are often too short to contain the testes, leaving the testes completely or partially ectopic. The right hemiscrota is often unformed, leaving the right testis completely ectopic, so the animal has a half a scrotal sac containing the left testis. On some individuals, the two hemiscrota are separate, essentially resulting in two scrotal sacs. On the 8 male white-tailed deer fawns examined from the 2007 fawn crop, all but one had the left hemiscrota formed directly forward of the right hemiscrota. The one bilateral scrotal sac was just a short bump with the testes horizontal under the skin.

There have been many studies of calves and lambs exposed to cyanogenic compounds in plants that describe similar skeletal malformations to those we have documented. Additionally,

the goats born to mothers deliberately given cyanide (Soto-Blanco and Gorniak, 2004) had elevated cholesterol levels, obesity and diabetes as a result of the cyanide caused thyroid hormone disruption. Possibly not a coincidence, those health problems in humans in Western Montana are reported to have tripled in incidence since we began seeing the health problems in newborn ungulate species in spring of 1995. There is also a high rate of both hyperthyroidism and hypothyroidism in adult humans. Over five million adults have been diagnosed with hypothyroidism in the U.S. As hypothyroidism often goes undiagnosed, this indicates that humans are likely being seriously affected by what is causing CFH in wild and domestic animals.

Most interesting, in a study of pregnant sheep given high levels of ethanol to simulate the effects of alcohol binge drinking by pregnant human mothers, both the mother and the fetus showed disruption to thyroid function (Cudd, T.A., Chen, W.-J.A., Parnell, S.E., West, J.R. Fetal and maternal thyroid hormone responses to ethanol exposure during the third trimester equivalent of gestation in sheep. *Alcohol. Clin. Exp. Res.* 26: 1065-1071, 2002), suggesting that Fetal Alcohol Syndrome (FAS) is the result of CFH. This would explain the close similarity of symptoms in human babies with FAS and symptoms of CFH in the animal young we have documented. Additionally, the ethanol exposure during the third trimester decreased fetal thyroid and thymus mass (Cudd, et al., 2002). This corresponds to the symptom of thymic aplasia that were found in individual newborns of both wild and domestic ungulate species and in RC calves with Weak Calf Syndrome (WCS) (Severson, per comm., 2006; Ward per comm., 2003). I autopsied animals that died at or soon after birth. Of 21 RC beef calves diagnosed with WCS, a newborn goat and several newborn deer fawns, all had maxillary prognathism, some with small eyes, in addition to little to no observable thymus. The underdeveloped eye openings documented on the calves and deer is a listed symptom of FAS and most of the above listed newborns had one or more of the other symptoms listed for both CFH in foals and FAS in human babies (Allen, 1996; CDC Website, 2007). However, while accident killed RC white-tailed deer older than three weeks of age had normal appearing thymus, many had symptoms of bone underdevelopment, including cranial/maxillary underdevelopment, maxillary brachynathia and narrowness between the eyes, with small eye openings and males had short and/or misaligned scrotal sac and/or on deer, short penis sheath. Again the variation in symptoms appears to be related to toxin combinations and timing of thyroid disruption to the maternal parent prior to or at conception and/or at various stages in development of the young after conception. Exposure just prior to or immediately after the egg is fertilized, when the first division of the egg cells is adversely affected, would likely result in the most severe damage, most often chromosomal damage, and frequently mortality during development.

Developmental malformations associated with fetal thyroid dysfunction have consistently increased in incidence in a large number of wild and domestic mammal species and has been reported or observed on individuals of many avian species throughout the United States. Many manufactured chemicals have the ability to alter the expression of thyroid hormone responsive genes. With proper amounts of thyroid hormone being essential for normal brain development in all vertebrates, including human children, the increasing rates of CFH related malformations should raise red flags regarding human fetal development. Many children have observable skull and maxillary underdevelopment and other symptoms associated with CFH, including premature birth, neurological cretinism, vision and hearing problems due to aplasia,

thymic aplasia, diabetes, obesity, heart disease, underdeveloped external genitalia and others. Additionally, the autism rate for children in Montana has more than tripled since 1994 according to newspaper reports.

Many of these developmental defects are associated with chromosomal damage, which many organochlorine pesticides (including Chlorothalonil) and other toxins have been shown to cause. For example, a large deletion from chromosome 22 during recombination at meiosis results in several genes from that region to be absent. This is referred to as DiGeorge Syndrome in human children. The many malformations incurred by damage to chromosome 22 is thought to be related to the amount of genetic material lost in the chromosomal deletion. It is significant that the DGS gene is required for the normal development of the parathyroid glands, thymus and heart, as underdevelopment of the thymus and maldeveloped hearts are commonly seen in the ungulate species with CFH. DiGeorge syndrome, diabetic embryopathy, fetal alcohol syndrome, Zellweger syndrome in human babies and CFH in all vertebrates have many distinct malformations in common, including failure of the thyroids, heart, thymus, eyes, ears and bones, especially the skull and facial bones, to develop normally. Congenital absence of the thymus results in deficient cellular immunity and susceptibility to infections, often resulting in mortality in young. Thymic aplasia in newborns also appears to be closely related to the development of autoimmune disorders later in life.

Chromosomal damage to Chromosome 13, 18 or 21 are called Trisomy in human newborns. According to attending obstetrics nurses, there were eight babies born to Western Montana parents the last week of July 1997 that had one of the three Trisomys, plus one baby with a heart malformation. This is quite a large cluster of malformed babies for Western Montana, with quite a variety of developmental problems. There have been many more malformed babies born since. There has also been a significant increase in premature births in Western Montana, as well as increasing rates of underdeveloped genitalia on male babies throughout the U.S.

Wild animals are often a sentinel for environmental problems that affect humans. It would seem imperative to immediately test the air, water and foliage to identify substances, especially Chlorothalonil and metabolites, that may be contributing to the alarming epidemic of CFH in wild and domestic vertebrates. Determined causes of CFH should be decreased or eliminated as soon as possible so the fetal thyroids on young of all species are able to function properly. All use of Chlorothalonil should cease until its role in causing CFH is determined.

A final observation is that it will take a very large number of people working together to instigate finding why the young of so many vertebrate species have such severe symptoms of CFH and instigate immediate changes to mitigate exposure to industrial hormone disrupting toxins, vehicle exhaust, inadequate sewage processing, open burning and spraying of hormone disrupting chemicals. It will be difficult for the next generation to pass on a better world to their children, if we do not immediately stop what is happening to the young of vertebrate species, including our children and restore their right to a normal development.